

### **AMENDMENTS TO THE SPECIFICATION**

Please amend the paragraph on page 13, lines 4-17 as follows:

Additionally, Clark, E. et al. disclosed that the treatments (such as gamma irradiation) that cause a loss of cell cycle control at the G<sub>1</sub>/S checkpoint cause HIV-1 infected cells to lose p21 function, and undergo apoptosis (Clark E et al. (2000) "LOSS OF G(1)/S CHECKPOINT IN HUMAN IMMUNODEFICIENCY VIRUS TYPE 1-INFECTED CELLS IS ASSOCIATED WITH A LACK OF CYCLIN-DEPENDENT KINASE INHIBITOR P21/WAF1," J Virol. 74:5040-5052). Gomez, T. *et al.* (<http://www.retroconference.org/2002/Posters/13446.pdf> [www.retroconference.org/2002/Posters/13446.pdf](http://www.retroconference.org/2002/Posters/13446.pdf); "CYTOPLASMIC P21<sup>WAF1/CIP1</sup> PROTECTS U937 PROMONOCYTIC CELLS FROM HIV MEDIATED APOPTOSIS") disclose that the administration of p21-antisense oligonucleotides to promonocytic cells suppressed p21 levels in the cells, and accelerated the death of the HIV-infected cells. The results are stated to indicate that p21 confers resistance to HIV-induced apoptosis in promonocytic cells, and to suggest a possible mechanism for the persistence of its infection in cells such as macrophages.